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# Delays in HIV Diagnosis Due to Differential Chorea Diagnosis Huntington's Disease Diagnosis by Years

**Janis Achen***

*Department of Neurology, Huntington Center North Rhine-Westphalia, Ruhr-University ,Bochum, Germany*

## Introduction

A CAG-trinucleotide repeat expansion in the huntingtin gene (HTT) is the root cause of the neurodegenerative Huntington's disease (HD), which results in a variety of diverse motoric, mental, and cognitive deficits . The vast hypotheses of underlying pathomechanisms and heterogeneous interactions have not yet been fully explored, despite the abundance of clinical symptoms. Numerous discoveries from preclinical and clinical investigations looked into a range of pathways in an effort to build models of the pathomechanisms underlying HD and identify potential treatment targets. The function of the huntingtin protein and its interactions with binding proteins (such as the ubiquitin-conjugating enzyme) as well as its part in many molecular processes, such as nuclear inclusions and the effect on cell death, were discovered. Additionally, the increased polyglutamine in HD causes aberrant protein-protein interactions, mitochondrial malfunction, excitotoxicity, and altered gene transcription, all of which decrease neuronal lifespan.In regards to increased cytokine production, an inflammatory response, and neurodegenerative processes, a clear connection between neurodegenerative processes and neuroinflammation has recently been found. As endotoxin- or microglia-mediated inflammation fuels neurodegenerative disease molecular pathways, it is becoming more and more clear that inflammation in neurodegenerative disorders is both a cause of and a result of neurodegenerative development. The innate and adaptive immune systems' inflammatory mechanisms are considered to be important HD pathomechanisms. To date, it has not been fully understood how neurodegeneration, neuroinflammation, and the expression of the clinical illness pattern are related. However, the primary goal of the recently completed clinical trial LEGATO-HD, which examined the impact of laquinimod in HD, was not met. HD, like Alzheimer's and Parkinson's disease, is characterised by CNS immune cell infiltrations, activated microglia, increased production of cytokines, and oxidative stress, along with other neurodegenerative disorders, neurovascular diseases, chronic pain conditions, diabetes, and HIV infection. This suggests, at least in part, similarities in the underlying mechanisms of the interaction and potential connections between neuroinflamm [1,2].

## Discussion

We have demonstrated, in accordance with an inspiring case report, that male HIV-positive patients had a longer diagnostic delay of nearly two years between the onset of symptoms reported by themselves and HD diagnosis compared to uninfected male HD patients. ENROLL-HD is the largest database of HD patients worldwide. Unexpectedly, people with both conditions did not have cognition damaged more severely.

In some circumstances, chorea's differential diagnosis may be difficult. We discussed a case of combined HIV/HD where there was a 7-9 year gap between the patient or family noticing any signs and the doctor diagnosing HD. The most frequent cause of generalised hereditary chorea is HD. If typical hyperkinetic movement problems are seen, the start of a moleculargenetic test of the mutant HTT might be recommended as a first step to prevent hazards associated with inappropriate diagnostic techniques. This strategy, in our opinion, may be particularly appropriate in the case of a broad chorea, whereas a hemichorea may be a sign of structural brain damage. When other movement disorders including myoclonus, dystonia, and hypokinetic rigidity predominate or the family history is ambiguous, as in our case report, the diagnosis may be more challenging. About 8–10% of HD patients reported having a negative family history, hence this does not rule out HD [3-6].

## Conclusion

In conclusion, we have demonstrated that in male patients with HIV and HD, there is a significant diagnostic delay of years between the onset of clinical signs and HD diagnosis. This delay may be explained by a blind spot in treating physicians who mistakenly attribute new symptoms to the known HIV infection. Therefore, even in the absence of a strong family history of HD, treating clinicians should be alert to consider potential other diagnoses if there is evidence of subcortical atrophy and a history of more generalised hyperkinesia. To prevent further pointless tests and enhance sociomedical care, those patients should be moved for early genetic testing.

## Acknowledgement

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## Conflict of Interest

None.

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***Address for Correspondence:** Janis Achen, Department of Neurology, Huntington Center North Rhine-Westphalia, Ruhr-University ,Bochum, Germany, E-mail: janisa@gmail.com*



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